Gastrointestinal complications after aortic surgery

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Background and Purpose: A major gastrointestinal complication (GIC) after aortic surgery may be disastrous, but these complications have received scant attention. This study was performed to determine the risk factors, associated events, and outcomes for patients with GIC.

Methods: We performed a secondary analysis of a prospective study that examined 120 consecutive patients who underwent transperitoneal aortic revascularization for aneurysmal or occlusive disease.

Results: The following 29 GICs developed in 25 patients (21%) within 30 days of aortic surgery: paralytic ileus that required replacement of nasogastric tubes (n = 12), upper gastrointestinal bleeding (n = 5), *Clostridium difficile* enterocolitis (n = 5), acute cholecystitis (n = 2), mechanical obstruction (n = 2), ascites (n = 2), and colon ischemia (n = 1). Seven patients required operations for GICs after aortic revascularization. A comparison of patients with and without GICs showed no differences in the prevalence of risk factors, presence of mesenteric artery stenoses, coexisting medical illnesses, antecedent gastrointestinal history, operative indication, preoperative fluid administration, or duration of operation. However, patients with GICs had more intraoperative complications (P = .004), greater intraoperative blood loss (P = .02), and more fluids during the postoperative period (P = .008). The mean duration of mechanical ventilation was 71 ± 23 hours for patients with GICs versus 7 ± 2 hours for patients without GICs (P = .006). A higher prevalence of pulmonary (P = .004) and renal (P = .001) complications was seen in the patients with GICs. The mean stay in the intensive care unit was 16 ± 2 days for patients with GICs as compared with 5 ± 0.4 days for patients without GICs (P < .001). Four deaths occurred, all caused by multisystem organ failure: 3 patients had GICs, and 1 did not have a GIC (P = .007).

Conclusions: These results show that GICs are prevalent in transperitoneal aortic surgery and are associated with severe morbidity rates, increased hospital costs because of prolonged stay, and increased mortality rates. Some GICs appear to be associated with intraoperative events that lead to visceral hypoperfusion, and others can be attributed to mechanical causes. However, none of the variables examined in this study were predictive of GICs. In all, GICs should be considered serious adverse sequela after aortic revascularization. Because no risk factors for GICs have been identified, these complications currently cannot be prevented. (J Vasc Surg 1998;28:404-12.)

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A postoperative gastrointestinal complication (GIC) may be disastrous and may result in severe morbidity, prolonged hospitalization, and even death.[1-5] GICs are particularly serious for patients who undergo operations remote from the abdominal cavity. The reported prevalence rate of GIC is approximately 2% after open cardiac operations,[1-3] 7% after neurosurgical operations,[5] 20% after cardiac transplantation,[3] and more than 50% after orthotopic lung transplantation.[4] Up to one half of the patients who have GICs require abdominal operations for

treatment,[2,3-5] and reported mortality rates range from 16% to 67%.[1,2,4,5] Although a number of possible risk factors for GICs have been proposed, visceral hypoperfusion appears to be the common pathologic mechanism underlying most of these cases.[6,7]

The incidence rate of GICs may be higher for patients who undergo abdominal operations, particularly for those who undergo transabdominal aortic operations for aneurysmal or occlusive disease. The risk of visceral hypoperfusion is omnipresent in these patients because of the risk of perioperative volume depletion and shock. In addition, associated visceral artery occlusive disease is common for patients who undergo abdominal aortic operations,[8] which may represent additional risk for hypoperfusion in some cases. Despite the potential importance of GICs for patients who receive prosthetic graft material, the impact of GICs on the results of aortic surgery remains unknown. Previous reports have focused on the prevalence and the outcome of specific complications, such as bleeding, bowel ischemia, and mechanical obstruction.[9-11] These complications are individually rare, but the associated morbidity and mortality rates are high for patients who undergo aortic operations.[9] When examined as aggregates, GICs may be shown to have serious adverse effects on aortic surgery results. We hypothesized that GICs are common sequela of transperitoneal aortic surgery and are associated with significant morbidity rates and increased hospital costs. The purposes of this study were to determine risk factors, associated events, and outcomes in patients who with GICs after aortic operations.

PATIENTS AND METHODS

This study represents a retrospective analysis in a group of patients on whom data was collected for a previous study unrelated to GICs.[12] The initial study was approved by the Institutional Review Board of the Dallas Department of Veterans Affairs Medical Center, and all patients who underwent elective abdominal aortic surgery for aneurysmal or occlusive disease were considered for enrollment. Patients with advanced cardiac disease (ie, unstable angina, myocardial infarction within 3 months, coronary revascularization within 6 weeks, severe aortic or mitral valvular disease, or clinically overt congestive heart failure) or advanced renal insufficiency (≥3.0 mg/dL serum creatinine level) were excluded. We also excluded infrequent patients who were undergoing redo aortic operations and patients in whom additional procedures (eg, femoropoliteal or renal artery bypass grafting) were performed.

Data regarding patient demographics, atherosclerotic risk factors, cardiac risk assessment, operative details, perioperative fluid administration, duration of hospital stay, and operative outcome were gathered prospectively. Medical records were reviewed retrospectively to determine a history of gastrointestinal disease (ie, peptic ulcer disease, hepatobiliary disease, ascites, gastrointestinal bleeding, intestinal obstruction, or pancreatitis). Previous laparotomies were noted. The duration of stay in the intensive care unit, the total hospital stay, and the duration of nasogastric suction were determined for each patient.

When available, aortograms were reviewed to determine the presence of stenoses in the celiac and superior mesenteric arteries. This presence was determined from lateral views of the upper abdominal aorta, which are a part of the routine aortographic procedure at our institutions. The assessment of the inferior mesenteric artery (IMA) flow was not considered in this study because routine injection of the IMA is not performed at our institutions and because overlying branches often obscure the evaluation of IMA patency. The method for calculation of the degree of arterial stenosis in the celiac and superior mesenteric arteries has been described previously.[8] For purposes of definition in this study, stenoses with a 50% or greater diameter loss were considered to be hemodynamically significant.

After aortic revascularization, the following GICs were tabulated: gastrointestinal bleeding, mechanical intestinal obstruction, ischemic bowel, acute cholecystitis, acute pancreatitis, enterocolitis, and adynamic ileus. Adynamic ileus was determined to be present if, after the removal of the nasogastric tube, a patient had gastric distention or vomiting that required tube replacement. The cause of the ileus was defined in all cases. A mechanical intestinal obstruction was defined as one which necessitated operative intervention to correct a site of intestinal obstruction within the first 30 days of surgery.

Stool guaiac determinations were determined routinely at the time of admission, and all results were negative for occult blood. The patients did not undergo preoperative mechanical bowel preparations. The operations were performed transperitoneally through anterior abdominal incisions. Intravenous first-generation cephalosporins were given routinely 30 to 60 minutes before the skin incision and continued for 48 to 72 hours after surgery. Patients with severe penicillin allergies were given vancomycin. Nasogastric tubes were placed at the time of initial abdominal exploration, and tube location in the distal antrum was verified by palpa-

Table I. Patient demographics and atherosclerotic risk factors

Characteristics	*No of patients (%)*
Age (years)	63 ± 0.8
AAA*	58 (48)
Race	
Black	8 (7)
Hispanic	2 (2)
White	110 (92)
Smoking	110 (92)
Hypertension	73 (61)
Hyperlipidemia†	48 (40)
Diabetes mellitus	18 (15)
Coronary disease	58 (48)

*Aneurysmal disease.
†Serum cholesterol >240 mg/dL or serum triglycerides >350 mg/dL.

Table II. Antecedant gastrointestinal problems

Prior GI problems	*No of patients*	*Previous laparotomy*
Peptic ulcer disease		
Pain only	12	5
Previous bleeding	8	2
Perforated	1	1
Hepatobiliary		
Cholelithiasis	5	4
Cirrhosis*	1	0
Colonic disease		
Lower GI bleeding	3	1
Diverticulitis	2	1
Carcinoma	3	3
Other		
Abdominal trauma	2	2
Liver abscess	1	1
Pancreatic cancer	1	1
Pancreatitis	2	0
Total	41	21

GI, gastrointestinal.
*Patient with cirrhosis also had cholecystectomy.

tion. The duration of nasogastric suction was determined by the resident caring for the patient in the postoperative period. In general, nasogastric tubes were removed when bowel sounds were active and when the nasogastric drainage was less than 400 mL in 24 hours, which was usually on postoperative day 5. All patients underwent routine postoperative gastroduodenal stress prophylaxis with histamine-receptor antagonists until normal alimentation was resumed. After aortic revascularization, the sigmoid colon was interrogated routinely with a continuous Doppler-scan probe on the antimesenteric border. The absence of a biphasic Doppler signal indicated reimplantation of the inferior mesenteric artery into the aortic graft.

Continuous data are expressed as the mean ± the standard error of the mean. Statistical comparisons between categorical parameters were made with the χ^2 test, and comparisons between groups of unpaired data were made with the Student *t* test. Differences were considered to be significant at the level of $P < .05$.

RESULTS

Fifty-eight patients (48%) underwent repair for infrarenal aortic aneurysm (AAA), and 62 (52%) underwent aortic reconstruction for occlusive disease. Three patients for AAA had reimplantation of the IMA at the time of AAA repair because of the absence of a biphasic Doppler signal on the antimesenteric border of the sigmoid colon at the conclusion of aortic revascularization.

The mean age of the 120 patients was 63 ± 0.8 years, and all were men. The patient demographics and the atherosclerotic risk factors are shown in Table I.

Prior gastrointestinal disease. Forty patients (33%) had a history of 41 gastrointestinal problems (Table II). Twenty-one patients (18%) had a history of peptic ulcer disease (PUD), including 19 with duodenal ulcer disease and 2 with gastric ulcers. Eight patients with PUD required gastric operations. Among the remaining 13, PUD was diagnosed with upper endoscopy in 8 and with contrast radiography in 5. Eight patients—6 with duodenal ulcers and 2 with gastric ulcers—had prior gastrointestinal bleeding, 2 of whom required gastric resection. Another patient had a previous duodenal ulcer perforation. None of the 21 patients for PUD had ulcer symptoms or signs of gastrointestinal bleeding at the time of admission. All ulcers were assumed to be healed.

Twenty-one of the patients (18%) had undergone previous laparotomies. As noted above, 8 patients with PUD had a history of gastric resection. Five patients had a history of colon resection for carcinoma (n = 3), diverticulitis (n = 1), or lower gastrointestinal bleeding (n = 1). Four patients had a history of open cholecystectomy. Two patients had prior laparotomies for intraabdominal trauma. One patient had undergone a pancreatoduodenectomy for carcinoma, and one patient had undergone drainage of a liver abscess.

Visceral artery disease. One hundred patients for the study had aortograms that were available for review. Twenty other patients underwent AAA repair without preoperative aortography. Lateral aortography showed hemodynamically significant

stenoses in 30 patients (30%). Twenty-three patients (23%) had a celiac artery stenosis with a 50% or greater diameter loss, 4 (4%) had a superior mesenteric artery stenosis, and 3 (3%) had stenoses in both arteries.

Gastrointestinal complications. Twenty-five patients (21%) had 29 GICs within 30 days of aortic surgery (Table III). The mean duration of nasogastric tube drainage for the 120 patients was 6 ± 0.5 days (range, 2 to 45 days; median, 5 days). After removal of the nasogastric tube, 12 patients had paralytic ileus and required tube replacement for continued gastric distention (n = 3) or vomiting (n = 9). Eight of these cases were attributed to infection (4 cases of urinary tract infection and 4 of pneumonia), and 4 were caused by electrolyte abnormalities (hypokalemia). Ileus resolved in 11 patients after the correction of the metabolic abnormalities or after the treatment of the associated infection, and 1 patient died of multisystem organ failure complicating pneumonia. Three patients with prolonged ileus had other gastrointestinal complications (eg, gastrointestinal bleeding, ascites, and *Clostridium difficile* enterocolitis).

Five patients had upper gastrointestinal hemorrhage caused by diffuse gastritis (n = 2), duodenal ulcers (n = 2), and gastric ulcers (n = 1). These diagnoses were suspected when blood appeared in the nasogastric-tube aspirate and were confirmed endoscopically. Two of these patients had a history of duodenal ulcer disease, 1 of whom had previous upper gastrointestinal bleeding. All 5 patients had some degree of hemodynamic instability—3 had a heart rate >110 bpm, and 2 had a systolic blood pressure <95 mm Hg. None of the patients required a gastric operation, but all 5 underwent a transfusion with at least 3 units of packed red blood cells (range, 3 to 6). Prolonged paralytic ileus associated with line sepsis also developed in 1 patient with diffuse gastritis.

Five patients had *Clostridium difficile* enterocolitis, with severe abdominal pain, fevers, and profuse watery diarrhea. These diagnoses were made on the basis of clinical signs and positive stool test results for *Clostridium difficile* toxins. Two patients underwent sigmoidoscopy, which showed mucosal pseudomembranes affecting the lower sigmoid colon in both cases. All 5 patients received cefazolin during surgery. The mean duration of perioperative antibiotics for these 5 patients was 3 days. One of the 5 patients underwent right hemicolectomy for cecal perforation. The other 4 had resolution of symptoms after 7 to 14 days of nonoperative management that included antibiotics and fluid resuscitation. Two of the 5 patients with *Clostridium difficile* enterocolitis had other GICs (eg, paralytic ileus and gastrointestinal hemorrhage).

Table III. Postoperative gastrointestinal complications

	No patients	*Laparotomy required*
Adynamic ileus	12*	0
Gastrointestinal hemorrhage		
Gastritis	2	0
Duodenal ulcer	2	0
Gastric ulcer	1	0
Enterocolitis	5†	1
Cholecystitis	2	2
Mechanical obstruction	2	2
Ascites	2	1
Colon ischemia	1	1
Total	29	7

*Three patients had other gastrointestinal complications.
†One patient had other gastrointestinal complications.

Acute calculus cholecystitis developed in 2 patients within 10 days of the original aortic procedure. Neither of the patients had evidence of cholecystitis at the time of aortic revascularization. This diagnosis was confirmed pathologically after cholecystectomy in both cases. One patient recovered uneventfully after cholecystectomy; the other died of multisystem organ failure.

Mechanical small bowel obstruction developed in 2 patients. One patient had a history of prior laparotomy. Nasogastric tubes were removed on postoperative days 3 and 4, respectively, and both were replaced because of vomiting. One patient underwent exploratory laparotomy on postoperative day 8 after signs of peritonitis developed. The second patient's diagnosis was confirmed on postoperative day 10 with contrast radiography, which was obtained because of persistently high nasogastric tube output. Both patients were found to have short segments of intestinal infarction caused by internal hernias associated with adhesions. After intestinal resection, 1 patient recovered uneventfully, and the other patient ultimately died of multisystem organ dysfunction.

New-onset ascites developed in 2 patients within 4 days of the original aortic operation. One patient with a history of alcoholic cirrhosis underwent reclosure of the abdominal incision when an ascitic leak developed on postoperative day 4. The ascites resolved with intensive medical therapy, and the patient was discharged on postoperative day 16. The second patient had a history of chronic lymphocytic leukemia and was noted to have prominent lymphatic tissue around the abdominal aorta at opera-

Table IV. Group comparisons

	GIC	*No GIC*	*P value*
No	25 (21%)	95 (79%)	—
Age (years)	66 ± 6	63 ± 9	.07
PAC*	15 (60%)	45 (47%)	NS
AAA	16 (64%)	42 (44%)	.08
Smoking	24 (96%)	86 (72%)	NS
Hypertension	17 (68%)	56 (47%)	NS
Diabetes	3 (12%)	15 (13%)	NS
Hyperlipidemia	7 (28%)	41 (34%)	NS
Antecedant GIC	11 (44%)	29 (31%)	NS
Prior laparotomy	5 (20%)	16 (17%)	NS
Mesenteric stenoses†	7 (35%)	23 (29%)	NS

GIC, Gastrointestinal complications; *PAC*, pulmonary artery catheters; *AAA*, abdominal aortic aneurysms.
*Patients randomized to receive pulmonary artery catheters.
†Among 100 patients undergoing preoperative aortography.

tion. Chylous ascites developed but ultimately resolved after placement of a peritoneovenous shunt.

Colon ischemia developed in 1 patient after aortobiiliac bypass grafting for AAA. The IMA was not reimplanted at the time of AAA repair because biphasic Doppler flow had been detected on the antimesenteric border of the distal sigmoid colon. Colon ischemia was recognized on postoperative day 11 when the patient had melena and became obtunded. He subsequently underwent left hemicolectomy, end transverse colostomy, and creation of a Hartmann's pouch. After a slow recovery, the patient was discharged on postoperative day 69.

In all, 7 patients required operations for GIC after aortic revascularization. These operations included open cholecystectomy in 2 patients with acute calculus cholecystitis, lysis of adhesions and intestinal resection in 2 patients with small bowel obstructions, colon resection in 2 patients with perforated colons (1 ischemia and 1 *Clostridium difficile* enterocolitis), and placement of a peritoneovenous shunt in 1 patient with chylous ascites.

Group comparisons. In a comparison of the 25 patients in whom GICs developed with the 95 patients in whom GICs did not develop, no statistical differences in the mean ages, operative indications, or atherosclerotic risk factors were seen (Table IV). GICs developed in 9 patients (16%) with AAA, which was not significantly different when compared with the 5 patients (8%) in whom GICs developed after aortic reconstruction for occlusive disease. The number of patients who received pulmonary artery catheters during the preoperative period was not significantly different between the 2 groups.

Of the 100 patients with available aortograms, 20 patients had GICs. Seven of the 20 patients (35%) with GICs had mesenteric stenoses. This was not statistically different when compared with 23 of the 80 patients (29%) with mesenteric stenoses in whom GIC did not develop (Table IV). When patients with enterocolitis, ascites, or intestinal obstruction (ie, infectious or mechanical GICs) are excluded, GICs that may have been related to visceral hypoperfusion developed in a total of 14 patients. Three (21%) of these 14 had mesenteric stenoses, which was not significantly different when compared with 27 of the 86 patients (31%) with mesenteric stenoses in whom GIC did not develop.

No significant differences were found between the groups for the mean volume of intravenous fluids administered during the preoperative or intraoperative periods (Table V). The mean operative times were nearly identical for both groups. However, the mean estimated blood loss was significantly higher for the patients in whom GICs developed ($P = .02$; Table V).

Seven patients with GICs (28%) and 7 patients without GICs (7%) had intraoperative complications ($P = .004$). These complications included the following: hypotension (systolic blood pressure <90 mm Hg for >5 minutes) in 4 patients with GICs (16%) and 3 patients without GICs (3%; $P = .02$), cardiac arrhythmias in 3 patients with GICs (12%) and 3 patients without GICs (3%; NS), and bronchospasm in 1 patient without a GIC (NS).

After surgery, the mean duration of ventilation was significantly longer for the patients with GICs ($P = .006$; Table V). Patients with GICs had significantly more fluid administered during the first 24 hours after surgery as compared with the patients without GICs ($P = .008$). Pulmonary complications developed in 6 patients with GICs (24%; 3 cases of pneumonia and 3 of adult respiratory distress syn-

Table V. Perioperative results

	GIC	*No GIC*	*P value*
Fluid administration (L)			
Preoperative	2.2 ± 1.2	1.7 ± 1.1	NS
Intraoperative	6.0 ± 2.3	5.7 ± 2.3	NS
Postoperative	5.4 ± 2.4	4.3 ± 1.7	.008
Operative time (min)	245 ± 93	243 ± 77	NS
Estimated blood loss (L)	1.6 ± 1.7	1.0 ± 1.0	.02
Duration of ventilation (h)	71 ± 226	7 ± 11	.006
Length of stay (days)			
ICU	16 ± 15	5 ± 2	<.001
Total postoperative	24 ± 20	10 ± 6	<.001
Death	3 (12%)	1 (1%)	.007

GIC, gastointestinal complications; *NS,* not significant; *ICU,* intensive care unit.

drome) and in 5 patients without GICs (5%; 3 cases of pneumonia and 2 of adult respiratory distress syndrome; $P = .004$). Acute renal failure developed in 4 patients with GICs (16%) and in 1 patient without a GIC (1%; $P = .001$). The mean length of stay in the intensive care unit was significantly longer for patients with GICs ($P < .001$), as was the total postoperative hospital stay ($P < .001$; Table V).

Four of the 120 patients in the study (3.3%) died within 30 days of aortic revascularization. All 4 deaths were attributed to multiorgan system failure. Three patients (12%) had GICs, and 1 patient (1%) did not have a GIC ($P = .007$).

DISCUSSION

The problem of postoperative GICs has been examined most extensively in patients who undergo cardiac operations. In a study of 4473 patients who underwent cardiopulmonary bypass graft surgery, Huddy et al[3] reported that the relative risk of a GIC developing was 1:249 after closed heart operations, 1:66 when the heart was opened, and 1:5 after cardiac transplantation. The risk is even higher for patients who undergo lung transplantation.[4] Overall mortality rates are approximately 25% for patients who undergo cardiac surgery, with higher death rates for patients who require abdominal operations.[1-7] Because GICs likely contribute to the development of multisystem organ failure in these patients,[13] the reported death rates may be greatly underestimated. The most common GICs after cardiac operations are paralytic ileus, gastrointestinal bleeding, and acute cholecystitis.[1,2] Many authors have reported the association between GICs and clinical scenarios associated with the following: cardiac arrhythmias, decreased end-organ perfusion, postoperative hypotension, prolonged cardiopulmonary bypass grafting, and sustained low cardiac output that requires inotropic pharmacologic support or intra-aortic balloon pump.[1,2,6,14] The common feature appears to be splanchnic hypoperfusion as a result of a low-flow state.

These study results suggest that GICs occur in 1 in 4 patients who undergo transabdominal aortic revascularization procedures. The most common types are similar to those seen in patients who undergo cardiac surgery. The GICs are individually rare, but the aggregate of all GICs in this study were associated with increased morbidity and mortality rates and overall hospital costs. Patients who were affected had a higher number of intraoperative events, postoperative complications, and deaths. The increase in hospital costs was evidenced by the longer duration of stay in the intensive care unit and in the total hospitalization for patients with GICs.

Patients in whom GICs develop after aortic surgery may share several features with patients undergoing cardiac surgery. The most prevalent types of GICs are similar in both groups. As is the case after cardiac operations, events leading to visceral hypoperfusion in this study increased the risk of GICs. Intraoperative hypotension and cardiac arrhythmias were more common for our patients with GICs. The estimated intraoperative blood loss for patients with GICs was also higher, and more postoperative fluid was administered in the postoperative period for these patients. This suggests that GICs tended to occur in patients who underwent more difficult operations that were associated with larger fluid shifts. However, it should be pointed out that the mean duration of operation was similar with and without GICs. These fluid statistics possibly reflected intraoperative complications and were only indirectly associated with GICs.

On the other hand, the increased intraoperative blood loss for patients with GICs was not reflected by an increased intraoperative fluid administration.

We acknowledge that GICs may have been in part caused by inadequate resuscitation in these patients.

Asymptomatic mesenteric stenoses do not appear to increase the risk of GICs, probably because the mesenteric circulation is well collateralized in patients who are affected. Although it is interesting to speculate that reimplantation of the IMA prevented GICs in 3 patients, the small numbers involved and the uncertain IMA circulation in this study population precludes meaningful analysis. Overall, proximal celiac and superior mesenteric stenoses do not appear to play a large role in the development of GICs.

Visceral hypoperfusion is not the only variable associated with GICs. Small bowel obstruction and chylous ascites most likely have an underlying mechanical cause, and *Clostridium difficile* has an infectious cause. In both cases of early small bowel obstruction, intra-abdominal adhesions caused internal herniation and bowel ischemia. This complication is rare after aortic surgery, and this study's results are in keeping with those previously reported.[10] We suspect that the patient with chylous ascites had a lymphatic leak in the abundant lymphatics overlying the aorta. As have others, we found the peritoneovenous shunt to be curative after unsuccessful conservative therapy.[15] *Clostridium difficile* enterocolitis is generally considered to be a complication of antibiotic use, and cephalosporins are frequently implicated.[16] We are unable to determine why these 5 patients had enterocolitis. We do note that all 5 patients received cephalosporin for a mean of 3 days. This was not different from the antibiotic regime used in the other 115 patients who did not have enterocolitis. The incidence rate of this complication (4%) is in keeping with the rate reported for patients who received antibiotics at the time of the general surgery procedures.[17] None of the 5 patients appeared to have had visceral hypoperfusion at any time before the onset of symptoms.

Should we have included *Clostridium difficile* enterocolitis in this analysis? This complication has not been included in previous analyses of GICs. However, it should be pointed out that the patients in this study who met the diagnostic criteria for *Clostridium difficile* enterocolitis were significantly ill. Four of the 5 patients who were affected required fluid resuscitation and had prolonged hospitalization times of 7 to 14 days; the fifth required a hemicolectomy. Thus this complication is associated with severe morbidity rates and prolonged hospitalization. It is an important GIC, even if a clear cause could not be identified beyond simple antibiotic use.

The 10% prevalence rate of paralytic ileus in this study is in keeping with previous reports. Published data from studies in which patients undergoing aortic surgery were randomized to transperitoneal versus retroperitoneal approaches suggest that the incidence rate of paralytic ileus ranges from 7%[18] to 10%[19] after transabdominal operations. The lower incidence rate of ileus after retroperitoneal operations reached statistical significance in both previous randomized trials.[18,19] Although we did not perform aortic revascularization with retroperitoneal approaches in the present study, the previous data suggest that retroperitoneal operations will significantly decrease the risk of postoperative ileus, and therefore the overall risk of GICs.

Despite being a relatively large analysis of patients who underwent aortic reconstruction, this study did have limitations. Because it is a retrospective analysis of data gathered from a prospective study, a number of GICs may have been missed. Nevertheless, the prevalence of individual GICs is in keeping with previous reports,[9-11] which suggests that the aggregate prevalence is within a reasonable range. We acknowledge that the definition of paralytic ileus may have been too strict in this study and that some patients with more benign ileus may have been missed. However, the 12 patients who met the current criteria for ileus showed a definite increase in morbidity rates and hospital durations. Although ileus was symptomatic of a more serious underlying complication in all instances, it served as a marker for worse outcome.

Another potential limitation of this study is that the nature of our referral population restricted the focus to men. Therefore our findings do not apply to women. Previous reports find no significant differences in prevalence rates of GICs between men and women who underwent cardiac procedures[2,3] nor in specific GICs for men and women who underwent aortic revascularization.[9-11] By extrapolation, we speculate that the aggregate risk of GICs is equal for men and women after aortic revascularization.

In conclusion, the findings of this study confirm that GICs are prevalent after transabdominal aortic revascularization and are associated with severe morbidity rates, increased hospital costs, and increased mortality rates. In some cases, GICs appear to be associated with intraoperative events that lead to visceral hypoperfusion. Other GICs may be attributed to mechanical causes. However, none of the variables examined in this study were predictive of GICs. Regardless of cause, GICs should be considered serious adverse sequela after aortic surgery. Because no risk factors for GICs have been identified, these complications currently cannot be prevented.

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DISCUSSION

Dr Calvin B. Ernst (Detroit, Mich). The authors are to be commended for bringing an important subject observation to our attention. Even though the study was retrospective and some historical marker data may have been overlooked, I agree with the final sentence in the manuscript that notes "regardless of cause, gastrointestinal complications should be considered serious adverse sequelae after aortic surgery."

Beyond noting that there were significantly more operative technical complications reflected by greater operative blood loss in the group with gastrointestinal complications than in the group without the complications, it would have been of more clinical significance if the authors could have identified risk factors that predict gastrointestinal complications. Then, preventive measures might have been taken analogous to the development of postoperative ischemic colitis where patients at risk can be identified and preventive measures taken, such as implantation of the IMA. Clearly, complications during operation lead to complications in the postoperative period.

My first question then asks if you could be a little more specific regarding the operative problems that may have eventuated in the gastrointestinal complications.

Secondly, did the gastrointestinal complications increase fluid requirements in subsequent pulmonary complications with increased need for ventilatory support and longer intensive care unit stays, or did the increased fluid requirements in pulmonary complications cause the gastrointestinal complications, most of which were episodes of paralytic ilias?

Among the 3 patients who died in the group with gastrointestinal complications according to the manuscript, it is unclear what the causes of the deaths were. I think you should be a little more specific about that.

In addition, what are the differences between the 2 groups in need for ionic tropic support during and after operation? Perhaps patients who developed gastrointestinal complications did so as a result of ionic tropic pharmacologic agents that caused varying degrees of splanchnic hypoperfusion.

Finally, this report begs to question whether there would have been fewer gastrointestinal complications had a retroperitoneal approach to the aorta been used. Certainly,

there may have been a decrease in the frequency of postoperative ileus, which accounted for about 40% to 41% of gastrointestinal complications.

Actually, I enjoyed this very well-presented paper, and I think it may have provided another marker, which, if predicted and prevented, may improve results of aortic reconstruction.

Dr R. James Valentine. We too were disappointed that we were unable to find any predictors of gastrointestinal complications in this study. I think this is probably a result of the multifactorial etiology of the complications in this series. As in the literature dealing with the cardiac surgery, there were a significant number of patients who had hemodynamic changes that were compatible with visceral hypoperfusion. Perhaps that is the major cause of gastrointestinal complications that might be preventable.

We do think that it is important to note that there were no differences in the risk of gastrointestinal complications among patients who had previous gastrointestinal disease, previous laparotomies, or mesenteric stenosis.

You asked whether patients who had gastrointestinal complications had increased fluids as a result of the gastrointestinal complications or whether increased fluids and blood loss were causative. It is difficult to say. I would suggest that the increased blood loss probably reflected longer operative time in many of the patients, but some were probably due to technical errors. There were some episodes of significant hypotension in these groups.

The causes of death were all due to multisystem organ failure. One patient died after cholecystectomy for acute cholecystitis, and another died after resection of infarcted intestine. Another patient who had a paralytic ilias actually died of a severe pulmonary infection, which developed into multisystem organ failure after severe pneumonia that was treated for 14 days unsuccessfully. The final patient died after colon resection for perforation from *Clostridia difficile* colitis.

The question of iontotropic support in the postoperative period is a good one. I do not have those data handy, but I would suspect that more patients with gastrointestinal complications probably required dopamine or some other type of inotropic support in the postoperative period. Of course, another possible issue is whether some of these patients required digoxin, and I again do not have those data for you right now.

The issue of whether we should have used a retroperitoneal approach is certainly controversial, and I think generally it remains to be seen and definitely proved whether a retroperitoneal approach really does reduce the prevalence of ileus after surgery. Some studies have suggested that it definitely does decrease the risk of paralytic ileus, and other have not found such a strong reduction in that risk.

We were able to identify, I believe, an etiology for every 1 of our patients in whom a paralytic ileus developed, with infection being the most common cause, usually pneumonia or urinary tract infection.